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# Clinical Proceedings

## OF THE CHILDREN'S HOSPITAL

WASHINGTON, D. C.

HISTOPLASMOSIS *Walter Pacosa, M.D., Chester Emmons, Ph.D.* ..... 95
THORACIC DUPLICATIONS OF THE ALIMENTARY TRACT *George William Ware, M.D.* ..... 103
FRACTURED AND DISPLACED PRIMARY AND YOUNG PERMANENT ANTERIOR TEETH *Rudolph J. Hursey, Jr., B.S., D.D.S.* ..... 106
THE USE OF HYPOTHERMIA IN CARDIAC SURGERY, INCLUDING A REPORT OF FOUR PATIENTS *John O. Nestor, M.D., Bernard J. Walsh, M.D., Edgar W. Davis, M.D., F.A.C.S., Charles E. Fierst, M.D.* ..... 108
EDITORIAL—EARLY REPAIR OF INGUINAL HERNIA *John Bailey, M.D.* ..... 114

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# HISTOPLASMOSIS

Walter Pacosa, M.D.
Chester Emmons, Ph.D.*

Histoplasmosis, up to a few years ago, was considered to be a rare, fatal disease. Increasing recognition of this mycosis has revealed that the majority of cases are benign and relatively asymptomatic. The protean manifestations of histoplasmosis make it necessary to consider this disease whenever pulmonary, mucocutaneous or gastrointestinal lesions are found in patients, especially those living or visiting in areas of high incidence. At present there seems to be an increase in the number of cases but this may be due only to more frequent recognition.

The object of this paper is to present a case of histoplasmosis and to review certain aspects of the disease of interest to pediatricians. It has been reported that approximately 30 per cent of the cases have occurred in the pediatric age group.

## CASE REPORT

J. N., a 12 year old white boy from Fauquier County, Virginia was admitted to Children's Hospital July 10, 1954 with a 3 week history of slight fever, cough, anorexia, vomiting after meals and malaise. He lost 12 pounds in the 3 weeks. At the onset of the illness, the family physician noted a sore throat and he prescribed 3 "shots" of penicillin. Three days before admission a diagnosis of pneumonia with "yellow jaundice" was made. Approximately 2 weeks before the onset of the illness this boy had cleaned a chicken coop at his grandfather's farm and deposited the debris in small piles of approximately one bushel each on a nearby field.

The past history was entirely negative with the exception of frequent occurrence of sore throats.

At the time of admission the boy's temperature was 101°, pulse 120, respirations 32, and weight 85 pounds. He was a well developed, poorly nourished, pale, chronically ill patient. No jaundice was visible. The lips were dry. The tonsils were normal but there was some pharyngeal injection. The tongue was coated. The heart was normal. The lungs were normal to percussion and auscultation. The abdomen was soft; no organs were enlarged. There were no enlarged lymph nodes. The admitting diagnosis was miliary tuberculosis or leukemia.

*X-ray findings:* X-ray examination of the chest on July 11, 1954 showed massive infiltrations from the apices to the bases, miliary in type, strongly suggestive of miliary tuberculosis. The hilar shadows were accentuated. (Fig. 1) Examination of the long bones showed no evidence of abnormality. A chest x-ray on July 26, 1954 revealed slight diminution in the miliary type of infiltration throughout the chest and there was some resolution of the density previously noted in the hilar areas. On August 31, 1954 there was still further resolution of the previously described process throughout both lungs. There was however, still some mottled infiltration throughout the chest. (Fig. 2)

* Head of Mycology Unit, Laboratory of Infectious Diseases, National Microbiological Institute, National Institutes of Health.

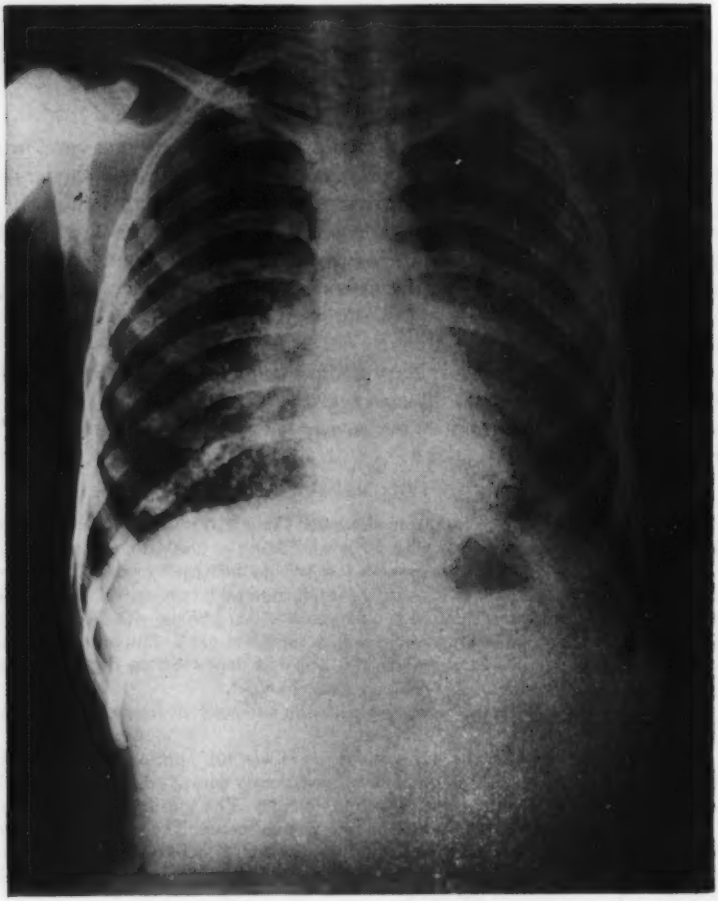

FIG. 1. "Miliary" pulmonary histoplasmosis

*Laboratory findings:* The urine was essentially normal with the exception of a sample taken July 26, 1954 which revealed 1 to 2 red blood cells per high power field and 10 to 12 white blood cells per high power field. Peripheral blood hemoglobin and white blood cell counts were normal. Eosinophilic cells constituted from none to 21 per cent of the leucocytes. Sedimentation rates varied from 46–32 mm per hour. The spinal fluid, Typhoid O and H, Brucella, Proteus OX-19 and heterophile agglutinins, total protein, A/G ratio, phosphorous, calcium, thymol turbidity, total bilirubin, and cephalin flocculation tests were normal. Complement fixation tests done at The National Institutes of Health with serums drawn on July 14 and August 2. 1954 were positive against a yeast phase Histoplasma antigen in a 1:4 dilution.

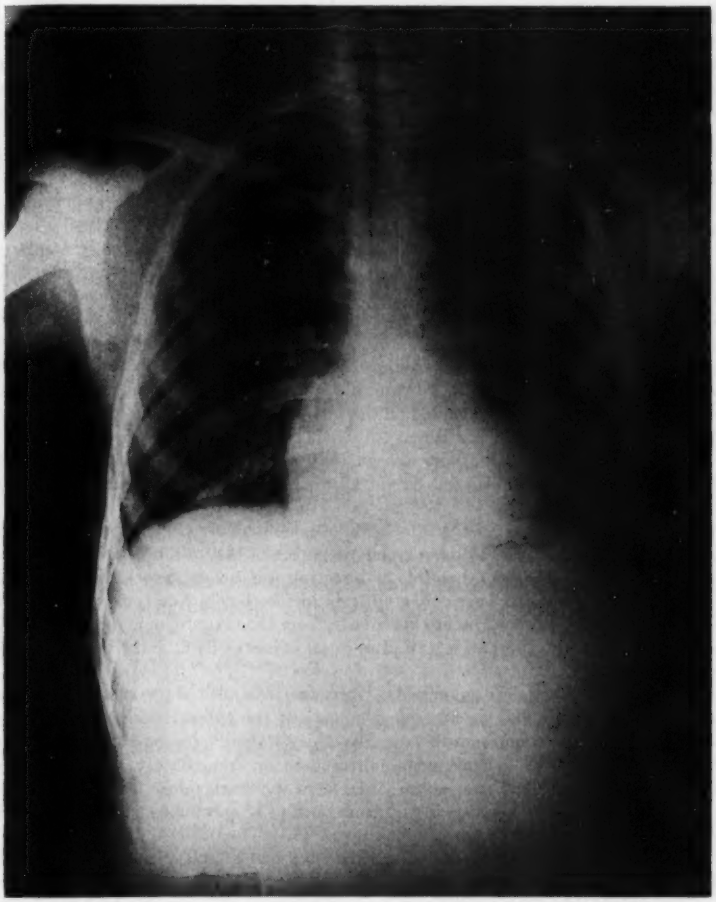

FIG. 2. Improvement in pulmonary histoplasmosis, 1½ months later

Complement fixation tests performed at Walter Reed Hospital on serums dated July 13 and August 20, 1954 reacted with Histoplasma 1:128 and Blastomyces 1:64 but were Coccidioides negative. Repeat complement fixation tests on September 8, 1954 were positive against Histoplasma 1:256 and Blastomyces 1:232; and negative against Coccidioides. Intermediate strength tuberculin tests (PPD) on July 10th and July 13, 1954 were negative, second strength PPD tests were negative on July 21st and August 2nd. Standard histoplasmin skin test on July 16, 1954 induced an erythematous area measuring 1 cm. in diameter with questionable induration. On August 2nd a repeat skin test caused an erythematous area 1.0 cm. by 1.5 cms. but no induration. The bone marrow on July 15th had an L:E ratio of 13:1, and a marked increase in

leukocytic elements, especially marked in the monocytic cells. Such a picture was considered compatible with tuberculosis. Bone marrow examination on August 12, 1954 showed an L:E ratio of 3:1 with an increase in immature granulocytes including eosinophiles; likewise reticulum cells were more numerous than usual. A right scalene node biopsy done on July 29, 1954 revealed hyperplasia of the reticulo-endothelial cells. Cultures of two bone marrow specimens, three gastric washings and the lymph node biopsy revealed no growth of *Mycobacterium tuberculosis* or *Histoplasma capsulatum*.

*Course:* Treatment with streptomycin, para-amino salicylic acid and isoniazid was instituted two days after admission. On the third hospital day slightly diminished breath sounds and vocal fremitus were noted over the left lung fields. Slight enlargement of an inguinal lymph node was also noted. Because of persistent vomiting, fluids were administered intravenously and para-amino salicylic acid was discontinued 5 days after admission. Non-productive cough and occasional headaches occurred and persisted. On the thirteenth hospital day, occasional temperature elevation to 100 and 101° developed and the liver enlarged 3 cm. to percussion. On the twenty-fifth hospital day the patient was afebrile and his appetite was improving. All three antituberculous drugs were discontinued on the fortieth hospital day. He was transferred to a convalescent home at this time.

On October 1, 1954 the patient was entirely asymptomatic. His weight was 105 pounds; physical examination was essentially negative. The sedimentation rate was 20.

*Probable source of infection:* On the basis of a compatible clinical course and supporting serologic reactions a presumptive diagnosis of histoplasmosis was made and a search for an environmental source of infection was begun. Specimens of chicken droppings, debris and soil were collected from inside the chicken coop which the patient had cleaned two weeks before his illness, from the manure he had removed from the coop at that time and had left in piles in an adjacent field, and from around the residence on this farm.

Fifty-one specimens were examined by injection into mice of the supernatant from a saline suspension of the specimen and culture of the spleen and liver of the mice when they were killed one month later. By this method[1] *Histoplasma capsulatum* was isolated from 9 of 17 specimens collected in or immediately adjacent to the chicken coop, from 2 of 17 specimens taken from the small piles of debris deposited by the patient in the adjacent field and from none of 17 specimens collected adjacent to the residence located on the same premises. These isolations of Histoplasma from an environment to which the patient had been exposed 2 weeks before onset of symptoms support the presumptive diagnosis of histoplasmosis.

## DEFINITION

Histoplasmosis is a severe, intermediate or benign fungus infection of the reticuloendothelial system. The severe form is a systemic disease which may involve almost every organ of the body while the benign form may manifest itself only as a mild respiratory disease. The clinical manifestations of systemic histoplasmosis are protean and include fever, cough, weight loss, ulcerations of the oro-pharynx and gastrointestinal tract, spleno-hepatomegaly and leucopenia.

### HISTORY

The first three recognized cases of histoplasmosis were reported from Panama by Darling[2] in 1906. He described the disease as being characterized by splenomegaly, emaciation and prolonged intermittent fever. In 1934 Dodd and Tompkins[3] reported the first case in an infant, a 6 months old child from Tennessee. DeMonbreun[4] first isolated the fungus from this case and described the cultural characteristics of the fungus. In 1945 Parsons and Zarafonetis[5] reviewed 71 cases of histoplasmosis, including 7 cases not previously reported. Eleven of the 71 cases were in children less than 1 year of age. In the Washington, D. C. area 4 fatal cases, 2 in siblings, were reported in 1946 from Loudoun County, Virginia by McLeod, Emmons, Ross and Burke.[6] In recent years the medical literature has contained numerous cases in both children and adults.

### ETIOLOGY

*Histoplasma capsulatum* is a dimorphic fungus which grows as a mold at room temperatures and as a yeast at 37° C. In the mycelial phase the fungus produces a white, cottony colony which becomes pale brown with the production of spores. Two types of spores are borne on terminal or lateral branches: microconidia which are spherical, 2.5–3.5 $\mu$ in diameter, with minutely spiny walls, and macroconidia which are spherical or occasionally clavate, 8–15 $\mu$ in diameter, with rather thick walls which are covered with finger-like appendages.

In the yeast phase Histoplasma is an egg-shaped cell, 2–3 x 3–4 $\mu$ in size which reproduces by budding at the smaller end. This is the form found in tissues and it can be maintained in vitro by incubating a culture on blood agar or other suitable medium at 37° C.

### INCIDENCE

The exact geographic distribution of histoplasmosis has not as yet been determined. It has been reported from 24 countries of the world. Within the United States most cases of histoplasmosis have been reported from the lower Mississippi Valley and central and eastern United States but the disease is not sharply delimited in distribution.[7] Within this area 20–60 per cent (85 per cent in some areas) of the general population react to the histoplasmin intradermal test. There is a high incidence of nontuberculous pulmonary calcification and histoplasmin sensitivity in an individual is generally interpreted as evidence of past infection with Histoplasma.[8]

### CLINICAL FEATURES

Discussion of the clinical manifestations may be divided into 3 categories related to the severity of the illness.[5] [9] [10] [11]

### 1. *Severe*

The majority of these cases occur in infants or the aged. The onset is insidious with failure to gain weight, cough, irregular elevations of temperature from 101° to 103°, malaise and indefinite joint pain. Loss of weight, cough and enlargement of the lymph nodes appear to be more prominent in the older children.

Affected children are apt to be irritable, pale and undernourished; rales and rhonchi may be noted in the lungs. There is an enlargement of the liver and spleen in the majority of these cases, especially in younger patients. Approximately 25 per cent show skin or mucous membrane lesions, oral lesions being more common in the youngest group and skin lesions in the older group. Anemia is seen in a majority of the younger group and approximately 25 per cent of the older patients. Bleeding abnormalities occur terminally.

### 2. *Intermediate*

In recent years there have been increasing reports describing a form of the disease intermediate in severity[12]. It occurs more commonly in the older child. In these cases there usually is a history of exposure to an environment such as chicken coops, cellars, barns or silos. Infection is accompanied by chills, fever, vomiting, cough and generalized aches and pains. There may be no abnormal findings other than slight splenomegaly. A mottled, generalized pulmonary infiltration is visualized by x-ray; in several months these lesions may show calcification. In the adult, various forms of chronic pulmonary infiltration, both with and without cavitation have been proved to be due to histoplasmosis. These cases have occurred rarely without symptoms. Diagnosis has been based upon skin sensitivity and complement fixation tests and has been verified with cultures in a few cases. There have been some reports which suggest that this disease may cause an atypical pneumonia-like illness. In some of these, development of calcification roentgenographically has been demonstrated at follow-up; in others skin sensitivity is the only evidence of this disease.

### 3. *Benign*

The majority of the cases of histoplasmosis are apparently benign[13-15]. Infection is revealed by cutaneous sensitivity to histoplasmin, occasionally accompanied by pulmonary calcification. This finding on x-ray cannot be distinguished from the primary tubercular infection; however, many of these patients do not react to tuberculin. Presumably the original infection is so mild that in many cases it cannot be recalled by the patient.

### LABORATORY DIAGNOSIS

Since a clinical diagnosis can not be made with certainty, laboratory confirmation is necessary. In many reported cases the laboratory diagnosis has been based upon skin sensitivity to histoplasmin and the demonstration of circulating antibodies. Both histoplasmin and the antigens used for complement fixation are nonspecific and the cross reactions with other mycoses, particularly blastomycosis, must be kept in mind when making a diagnostic interpretation. Skin tests and serologic tests should be made as early as possible in the course so as to show acquisition of sensitivity and a rise and fall in titre of circulating antibodies in subsequent specimens. Since serologic and dermal diagnostic tests are equivocal it is highly desirable to demonstrate the fungus in pus, ulcer base, blood, bone marrow smears, or in biopsies of tissue, and to isolate the fungus in culture from such sources. The Giemsa and periodic acid-Schiff stains are suitable for direct demonstration of Histoplasma. Cultures from uncontaminated material can be planted on Sabouraud's agar or blood and from contaminated specimens upon Sabouraud's agar to which 20–100 units per ml. each of penicillin and streptomycin have been added. Cultures should be incubated at room temperature or at 30° C and held for one month before discarding as negative. Cultures can be isolated in the yeast phase if blood agar tubes are incubated at 37° C but this is not a dependable method of isolation.

### DIFFERENTIAL DIAGNOSIS

Because of the various clinical pictures, the differential diagnosis includes many entities. In the pediatric age group, the presence of fever, anemia, hepatosplenomegaly and leucopenia lead to the consideration of aleukemic leukemia, lipoid storage disease, Banti's syndrome, infectious mononucleosis, tuberculosis and brucellosis. The presence of lymph node enlargement might lead to a diagnosis of Hodgkin's disease, sarcoidosis, and malignant lymphomas. If the picture is limited to the pulmonary system, primary atypical pneumonia, coccidioidomycosis and tuberculosis must be kept in mind especially in the adult and the older child. Cutaneous and oropharyngeal lesions of histoplasmosis may simulate syphilis.

### TREATMENT

At the present time there is no fungistatic or fungicidal agent which clearly influences the clinical course of histoplasmosis without toxic reactions. Many drugs, including penicillin, streptomycin, broad spectrum antibiotics, iodides, mercurials and stilbamidine have been tried with little success. Pulmonary lesions will usually heal satisfactorily with general supportive therapy. There is no need for anti-fungus treatment unless the disease is overwhelming.

SUMMARY

A case of febrile illness is reported with the presumptive diagnosis of histoplasmosis based on the presence of miliary lesions throughout both lung fields, dermal sensitivity to histoplasmin, circulating antibodies against Histoplasma, a suggestive history of exposure to litter from an old chicken house and isolation of *Histoplasma capsulatum* from the chicken manure and litter remaining in this chicken house and from one of the piles of litter which the patient had removed from the chicken house and deposited in a nearby field 2 week before the onset of his illness.

The epidemiology, diagnosis and attempted therapy of histoplasmosis are briefly reviewed.

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# THORACIC DUPLICATIONS OF THE ALIMENTARY TRACT

George William Ware, M.D.

Duplications of the alimentary tract have been defined as "spherical or tubular structures which possess a well developed smooth muscle layer and are lined with a mucous membrane. They are found at any level from tongue to anus and usually are intimately attached to some portion of the alimentary tube".[1] Duplications which are thoracic in location are described in this paper. Less than ninety such cases have been reported in the literature.[2]

## ETIOLOGY

This entity is congenital in origin and the embryological background forms highly interesting reading. Several theories have been proposed.

*Duplication:* Bremer is the principal exponent of this theory.[3] This author believes that duplication occurs in the embryo of six to eight weeks. At this stage epithelial cells are increasing more rapidly than the length of the alimentary tract. The excess cells fill the lumen of the gut. The secretions of these cells cause vacuoles which coalesce with each other and with the lumen. With continued growth of the intestinal tract, the excess cells migrate to form the wall of the gut. Normally the vacuoles open into the lumen and finally disappear. If a row or chain of these vacuoles persists, a duplication of the alimentary tract occurs.

*Diverticular Theory:* Lewis and Thyng have shown that intestinal diverticula are found normally in the embryo of man.[4] Such diverticula disappear in the normal course of embryologic development; abnormal persistence, either attached to adult alimentary tract or detached from it, would result in the lesion under discussion.

These two theories are the customary explanations given. Other less well accepted theories hold that a portion of primitive gut becomes detached and later undergoes development; a portion of the omphalomesenteric duct persists into post-natal life. These conditions may have an origin similar to dermoids or teratomas.

## *General Statistics*[2]

The majority of patients diagnosed are in infancy. The age of reported patients however has varied from still-born fetuses to adults in their fifties. Males are more commonly affected. The right hemithorax is more commonly involved. The mass however may project into both pleural cavities or extend across the diaphragm. Transdiaphragmatic duplications may originate in the abdominal cavity yet cause predominately thoracic symptoms.

### *Symptoms and Signs*

The most frequent symptoms reported were dyspnea, cough, cyanosis, vomiting and poor weight gain. Occasionally repeated pneumonia and/or chest pain were noted. Rare symptoms included hemoptysis, hematemesis, abdominal pain or hoarseness. In an occasional case the patient has no symptoms; the lesion in this instance is discovered on a routine chest film.

Physical examination is not diagnostic and frequently is entirely negative. When physical findings are present they are those of consolidation or a solid mass in the chest.

### *Radiography*

Plain chest film will reveal a well defined mass in the posterion mediastinum in the majority of instances.

### *Associated Anomalies and Disease*

A large number of patients will have concomitant orthopedic abnormalities such as hemivertebrae, spina bifida, scoliosis, or club foot. The reason for such association is unknown. Various other congenital anomalies may occur; abdominal duplications, meningocele, and congenital heart disease.

The most important associated disease is that of peptic ulcer. Actually this is a complication since it occurs in duplications which have a gastric mucosal lining in whole or in part. Ulceration will frequently lead to hemorrhage and perforation. The latter catastrophy often leads to abnormal communication with the pulmonary tree resulting in severe and sometimes fatal syndromes.[2]

### *Treatment*

Radiation therapy and thoracenteses have been used for this condition. Both methods have proved fruitless. Aspiration may be dangerous because of the danger of leakage of acid contents.

Surgical excision is the best treatment. Earlier forms of surgery consisted of marsupialization of the mass. This latter maneuver is not curative.

Concomitant pulmonary resections are necessary in cases of associated pulmonary suppuration secondary to peptic ulceration.

### *Pathology*

On examination of the specimen a definite smooth muscle component is seen. The mucosa will vary in histology. Gastric mucosa is most common, but may vary from esophageal to colonic. As with abdominal duplications, the mucosa need not conform with that of the level at which the duplication is found. There may be an admixture of types found. For example gastric,

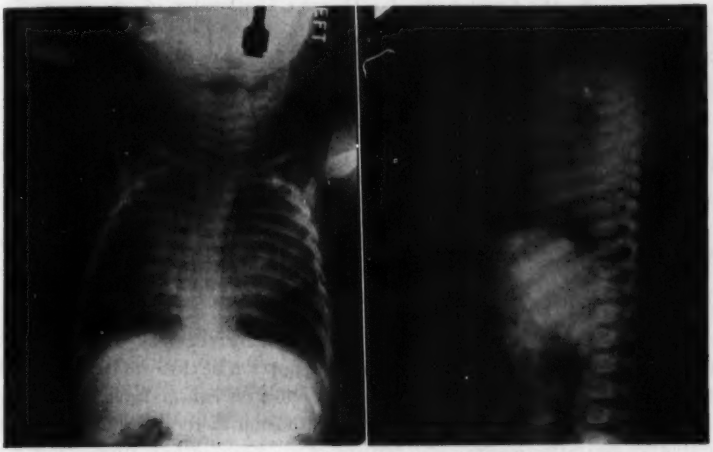

Fig. 1. X-rays showing thoracic duplication of the alimentary tract

esophageal, and enteric mucosa may be identified in the same surgical specimen.

CASE REPORT*

A male infant, aged 3 months, was admitted to Valley Forge Army Hospital because of a tumor in the left upper lung field. The patient was originally admitted to a hospital in Germany because of phimosis. During the admission examination cough and fever were noted and a chest film was obtained. When an abnormal haziness of upper left lung field was reported, the patient was transported to this country.

Physical examination revealed dullness to percussion in the upper posterior half of the left lung. Laboratory work was within normal limits. Radiographic interpretations were as follows: "Films of chest show a blunt shaped cone with its base against the posterior gutter on the left. The mass measures 5 centimeters across the base, 5 centimeters in height. The apex is round and the margins are sharply demarcated." (Fig. 1)

A left thoracotomy was performed. A large, well encapsulated mass was found in the postero-lateral portion of the left thoracic cavity. The mass was approximately 6 by 8 cms. in size and the mass was adherent to the lung. There was no connection with the esophagus. The mass was removed by sharp and blunt dissection.

On pathologic examination the mass was found to be a unilocular cyst. The mucosal lining was mixed with gastric and enteric cells. In the wall there were focal areas of pancreatic tissue.

The post-operative course was uneventful.

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## FRACTURED AND DISPLACED PRIMARY AND YOUNG PERMANENT ANTERIOR TEETH

Rudolph J. Hursey, Jr., B.S., D.D.S.

Due to the increasing complexity of our modern society there is an ever growing number of injuries to the anterior portion of the oral cavity, involving the anterior teeth. Such accidents in children can be most tragic, since these teeth are not only utilized for the incision of food, but are of inestimable value esthetically. The patient with such injury presents a most difficult problem for the dentist for many reasons.

Of prime importance is the problem of infection and the prevention of its spread. Since the veins of the head have no valves, a cavernous sinus thrombosis could follow. Other problems are the disagreeable attitude of the patient due to the injury received; the difficulty of restoration; and the realization that the entire future of the young person may be influenced by the outcome of the treatment and the positive attitude of the dentist. A restoration that is not functional or esthetic may lead to the development of an emotional aberration which could be a positive factor in determining the happiness and success of the individual. There are many individuals who have not attained their goals due to a faulty manner of speech, an acquired habit, or by their appearance as the result of such an injury. These several problems associated with traumatized anterior teeth must be approached with a great sense of responsibility.

The young child is very vulnerable to a fracture or displacement of the anterior primary teeth. These teeth are displaced more frequently than fractured because of the comparatively slight degree of calcification of the supporting bone. The pulp of these teeth has great recuperative power and the prognosis of such displaced teeth depends upon the general condition of the patient and the direction of injuring force. If the fulcrum of the displaced tooth is at the apex, the prognosis is favorable; however should the tooth be forced apically into the alveolar process or displaced bodily anteriorly or posteriorly, the prognosis is not so good. Survey of the records of the dental clinic at Children's Hospital indicates that in over 100 cases of traumatized deciduous teeth, 70 per cent remained vital with supportive

therapy and readjustment of the tooth with finger pressure. If these involved teeth become non-vital and turn dark but there is no active infection present, it is our policy to leave them in the arch and follow their status with frequent x-ray examination.

The condition of the mouth at the time of eruption of the permanent anterior teeth does not offer much protection from injury. These teeth are not well supported, since their root formation will not be completed for several years and therefore can easily be displaced. They are prominent in the mouth and usually do not have adequate lip coverage, thus leaving these teeth unprotected.

From the dental viewpoint there are several plans which may be instituted in the treatment of fractured and of displaced primary and young permanent teeth. Most of these procedures depend upon the amount of time that has elapsed since the accident occurred, this frequently being the all-important factor in the success or failure of the instituted treatment. The physician, therefore, after making his examination and carrying through any general medical therapy he deems necessary, should impress upon the parent the importance of the time factor in treating dental injuries. An exposed pulp, seen by the dentist within the hour of trauma, can usually be treated with far less radical procedures than one of several days' duration when associated pathology has developed. In those severe cases where there is central nervous system damage, a warm bland mouth wash should be used, if possible, until dental therapy can be undertaken.

In those cases where the young permanent anterior teeth are completely displaced from the arch, the tooth should be placed in benzalkonium chloride and the patient referred to his dentist immediately. It is advisable to give the patient prophylactic antibiotic therapy before leaving the physician's office. This would expedite matters greatly and would gain for the dentist valuable time.

The importance of extra-oral x-rays cannot be over-emphasized when the injury involves the mandible. In the 6 year old child, the mandible contains 10 deciduous teeth, the tooth buds of 10 succedaneous teeth, 4 permanent molars, 4 developing tooth buds of the second permanent molars, for a total of 24 teeth. These structures and the mandibular canal leave the horizontal mandible with little more support than the cortical plates of bone.

In conclusion, injuries to the anterior teeth present a real problem and should be handled as quickly as possible. There are cases where the root of the tooth is fractured with no external evidence. The fracture may connect with the oral cavity and be a pathway of infection. The physician can be of tremendous help by instituting any therapy he deems advisable before the patient leaves his office and impressing upon the parent the value of time.

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## THE USE OF HYPOTHERMIA IN CARDIAC SURGERY, INCLUDING A REPORT OF FOUR PATIENTS*†

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At the present time there are several technics for closed or indirect intracardiac surgery in which maneuvers within the heart are done blindly, the surgeon being guided only by the feel of the instrument or the finger. Examples of the nonvisual approach in cardiac surgery include commissurotomy for severe mitral stenosis by means of finger fracture or special knife, and incision of stenotic pulmonary or aortic valves by valvulotomes inserted through the heart walls. These procedures, though not ideal, are quite satisfactory for the partial relief of these conditions. However, in large congenital defects of the auricular or ventricular septum it is only by the development of a surgical technic which permits opening the heart and closure of the defect under direct vision that we can expect to benefit the patients having such defects. Certain patients who have, in addition to a ventricular septal defect, narrowing of the outflow tract of the right ventricle either at the infundibulum or the pulmonary valve and a dextroposed aorta over-riding the septal defect would be better treated by removal of the infundibular or valvular obstruction under direct vision than by the creation of an aorto-pulmonary shunt. This last group, all of whom are cyanotic, is classed under the heading of tetralogy of Fallot.

The first method suggested to improve upon these technics was the con-

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struction of a mechanical heart[1–8], a pump to furnish the driving force for blood in association with an oxygenator which might be the patient's lungs, a homologous lung, or a mechanical device. It was thought that the mechanical heart would make possible the temporary removal of the heart from the circulation for a period long enough to permit intracardiac surgery under direct vision. The technical difficulties associated with the manufacture and use of such an extracorporeal heart have been of such great magnitude as to make this appear an impractical device at this time.

A very recent and promising approach to direct surgery of the open heart has come about through the work of Lillehei and his associates[9] at the University of Minnesota. This group has used the heart and circulation of a normal individual connected to the patient by peripheral artery-to-vein hookup with a simple mechanical pump interposed between the patient and the normal individual. This was permitted the isolation of the patient's heart in a bloodless field and the successful repair of intracardiac defect in several patients.

Another approach, largely developed by Swan and his group[10, 11] at the University of Colorado, permits the opening of the heart and the repair of cardiac defects under direct vision. This is based on the knowledge that cooling of the body lowers the metabolic rate, reduces markedly the demand for oxygen by the tissues, and simultaneously enables the blood to carry more oxygen both in physical solution and as oxyhemoglobin. In fact, the oxygen requirement at 20° C is only about one fifth that at the normal temperature of 37° C. This is particularly important in those with cyanotic congenital heart disease.

Rather extensive experimental and clinical evidence indicates that hypothermia permits such surgery without extensive risk. For 150 years data[12] have been accumulated on the effects of hypothermia, chiefly from animal experiments, but occasionally from experiments on human beings, such as those reported in 1940 by Smith and Fay[13, 14] on patients with far-advanced malignant disease, and those performed by the Nazis on concentration camp inmates during World War II.

It was in 1942 that many of the physiologic effects on human beings of rapid cooling even to the point of death were determined under the direct order of Heinrich Himmler, the Nazi leader. In these experiments,[15, 16] which resulted in approximately 100 deaths, more than 300 experimental subjects were used in about 400 experiments. The problems studied included the treatment of persons who had been exposed by shipwreck to cold water for long periods of time, and the possible physical and pharmacologic methods of attack. It was hoped that the problem of the mechanism of death from cold would be solved. It was stated that it was especially difficult to carry the findings in animals over into the human field, since in the warm-

blooded animals there is a varied degree of development in the temperature-regulating mechanism and also the processes in the skin of the pelted animal could not be carried over to man. During these experiments fairly complete physiologic studies were carried out with respect to changes in blood, urine, spinal fluid, skin temperature, muscle tonus reflexes, and heart action. The effects of various pharmacologic and physical agents were also determined.

The data derived from the Nazi experiments on human beings indicated that death from cooling is primarily a cardiac death, and that the hitherto puzzling phenomenon of post-rescue collapse and death was the result of the so-called after-drop in temperature which occurred when the victims were removed from cold water. The most effective method of resuscitation in such cases was immersion in hot water to 45° C, which prevented this lethal after-drop and warmed the individual quickly. It was also determined that the commonly used stimulants had no obvious beneficial effect and that alcohol, contrary to common belief, actually hastened cooling, but there were indications of a possible protective action from alcohol on the heart.

It is hoped that additional valuable data will be uncovered as the result of a thorough search of existing captured documents that have not yet been reviewed and evaluated in the light of our present knowledge, and from interviewing medical personnel now known to have personal knowledge of this type of experimentation. Undoubtedly more lives can be saved by this information than were originally sacrificed to obtain it.

After the War, independently and without the benefit of detailed knowledge of the wartime experiments on human beings by the Nazis, Swan, Virtue, and others[10, 11] at the University of Colorado School of Medicine in Denver, as the result of biochemical research, developed a practical technic of direct surgery of the human heart with hypothermia. Swan's method has been used satisfactorily by others, including our own group, and it is this technic that we shall describe.

## METHOD

The patient is anesthetized in the usual fashion after which an endotracheal tube is inserted and connected to a closed anesthesia system to permit complete control of respiration by the anesthesiologist. The electrocardiographic electrodes are attached to the extremities, and an oscillograph is plugged into the electrocardiograph. The bulb of a regular commercial deep-freeze thermometer is inserted into the rectum. The patient is then immersed in a tub of ice water at from 2° to 12° C with only the head and neck protruding. From this moment on the anesthesiologist produces controlled hyperventilation to prevent acidosis and a change in the blood potassium level, and the cardiologist watches the electrocardiogram for the appearance of arrhythmias or cardiac arrest. A surgeon stands by com-

pletely prepared immediately to open the chest and institute appropriate treatment in the event of ventricular fibrillation or cardiac arrest.

When the temperature sinks to 30° C (86° F) the patient is lifted out of the ice water onto the operating table and placed on a rubber mattress through which water of controlled temperature is circulated. Ordinarily there is an after-drop of approximately 4° C before the rectal temperature stabilizes at about 27° C (80.6° F), which is considered the optimum for surgery. The heart rate slows to about 40 a minute, and the anesthesiologist maintains the respiratory rate between 40 and 60 a minute. The patient receives only 100 per cent oxygen, since anesthesia from the cold alone is adequate at this level.

The surgeon then makes a bilateral transverse sternal intercostal incision in the fourth interspace, since it is essential that all sides of the heart, the venae cavae, the ascending aorta, the pulmonary artery, and the lung roots be reached. Inflow of blood into the heart is stopped by occluding the venae cavae, and about a minute is allowed for the heart to empty itself and the pulmonary circuit as completely as possible. A non-crushing clamp is placed across the base of the pulmonary artery and aorta in a position deliberately to occlude the orifices of the coronary arteries. The slowly beating heart is then opened to allow the residual blood to escape and air to enter. The operative procedure is performed within a maximum time limit of 15 minutes. Just before the incision in the heart is closed, the chest cavity is rapidly flooded with Ringer's solution until the entire heart is submerged. As the heart beats, the air escapes through the incision, leaving its chambers filled with the solution. A second non-crushing clamp is then applied to the cardiotomy incision closing the heart. The clamp across the pulmonary artery, aorta, and coronary orifices is now removed. The superior vena cava is released and blood flow is allowed to resume. After 30 to 60 seconds, when the heart is observed to resume its work load, the inferior vena cava is released and total circulation through the heart is restored. The cardiotomy wound is closed and the chest wall sutured in the usual fashion.

The patient is then lifted from the operating table and immersed in a tub of hot water at 45° C. The rectal temperature slowly rises to 35° C (95° F), at which point the patient is taken from the tub and returned to his bed.

## REPORT OF CASES

*Case 1.* J. C., an 8-year-old boy with known cyanotic congenital heart disease, was first seen in the Cardiac Clinic of Children's Hospital on July 27, 1953. A diagnosis of tetralogy of Fallot was established and cardiac surgery recommended. On March 14, 1954 hypothermia was induced, and when the rectal temperature was 27° C (80.6° F) a transverse sternum-splitting thoracotomy was performed. The temperature was maintained at this level as the circulation was totally occluded for 6 minutes, 10 seconds, while the right ventricle was opened and an infundibular membrane excised

under direct vision. He was returned to his bed 7 hours after the start of anesthesia with normal temperature and when he had reacted sufficiently to talk to his mother. During the immediate postoperative recovery period a monoplegia with particular weakness of the right hand was noted. This has responded well to physiotherapy. There has been marked improvement in his ability to exercise with much less cyanosis and a 5-pound weight gain in the first 3 months after surgery.

*Case 2.* Y. C., a 4-month-old girl, was admitted to the Medical Service of Children's Hospital on May 1, 1954 because of an episode of syncope and cyanosis. Examination revealed delayed development, moderately severe generalized cyanosis, a grade 2, short, rough systolic murmur along the upper left sternum, a distinct, pulmonary second sound but without splitting, and easily palpable femoral pulsations. X-ray, fluoroscopic, and electrocardiographic examinations showed moderate enlargement with hypertrophy of the right ventricle, greatly diminished pulmonary vascularity, and scant evidence of the pulmonary artery or its main branches. A diagnosis of tetralogy of Fallot with severe pulmonary stenosis was made, and in view of the seriousness of the child's status early operation under hypothermia was recommended.

On April 4, 1954 anesthesia was inducted with nitrous oxide and ether and an intratracheal tube inserted for closed anesthesia. The child was immersed in ice water and within 1 hour the rectal temperature had fallen to 30° C (86° F). At the time of the initial skin incision the rectal temperature was 26.8° C. After operative exposure was complete it was evident that the patient had a small main pulmonary artery which ended blindly in the wall of the right ventricle. Under direct vision the right ventricle was opened and a cataract knife was introduced from the right ventricle through a 5 mm. thick ventricular wall into the lumen of the pulmonary artery. This incision was enlarged by the use of a small scissors. As the ventricular incision was closed unavoidable anesthesia difficulty developed, which shortly resulted in the patient's death on the operating table.

Autopsy revealed a large hypertrophied right ventricle, a very small thick-walled left ventricle, a high interventricular septal defect, dextroposition of the aorta, and a clean surgical opening from the right ventricle into the pulmonary artery, just at the site of the anterior commissure of the pulmonary valve.

*Case 3.* B. M., a 4-year-old girl with a known congenital cardiac defect, was first seen in the Cardiac Clinic at Children's Hospital during October, 1953. A tentative diagnosis of an auricular septal defect was made with a recommendation for cardiac catheterization. On December 20, 1953 she was admitted to the hospital in congestive heart failure with considerable dyspnea, a large liver, and anasarca. She responded rapidly to treatment, with disappearance of all edema and liver enlargement. Cardiac catheterization was carried out in the Cardiac Laboratory on February 16, 1954, revealing increased pressure on the right auricle and right ventricle and a 3-volume rise in the oxygen content of the blood in the right auricle as compared to the blood samples taken from the venae cavae. During this procedure the catheter was passed through a defect in the auricular septum into the left auricle.

On May 11, 1954 hypothermia was induced as previously described, and the right auricle was opened. A 5 cm. elliptical opening crossed by trabeculations was found in the auricular septum. This was closed by 6 sutures. The heart and circulation were occluded for 7 minutes and 40 seconds, the right auricle being open 6 minutes and 40 seconds of this period. Her recovery was completely uneventful, and her activity pattern subsequent to discharge from the hospital has been that of a normal child of her age.

*Case 4.* R. H., one of identical twins, was first seen by one of us (B. J. W.) at the age of 3 weeks. He had a weak cry, was feeble compared to his normal twin, and became cyanotic with hard crying. There were good pulsations in both femoral arteries. There was a slightly loud, high-pitched systolic murmur along the upper left sternum. The pulmonary second sound was distinct but not definitely split. X-ray, fluoroscopic, and electrocardiographic examinations suggested dextrocardia of the non-mirror image type, but with added unusual features concerning the heart chambers and great vessels which could not be clearly explained. It was evident at this early date, however, that there was a marked reduction in pulmonary circulation.

Occasional observations over the next 5 years revealed increasing generalized cyanosis, an exercise tolerance well below that of the normal twin, and occasional squatting. The heart size and shape and electrocardiographic features did not change materially. Cardiac catheterization was performed on October 20, 1953 but was incomplete and unsatisfactory. On May 27, 1954 an angiocardiogram revealed a right aorta which filled from the right ventricle and descended on the right of the spine. There was poor filling of the pulmonary artery and its branches.

On June 8, 1954, thoracotomy was performed under hypothermia. At the time of the initial skin incision the rectal temperature was 26.5° C (79.7° F). Complete exposure of the heart and major blood vessels revealed unusually small, non-pulsating pulmonary arteries. These vessels were well filled with blood but had no evident communication with either ventricle. The pulmonary arterial blood apparently came from an extensive collateral bronchial artery circulation. It was thought that no surgical procedure could be carried out at this time that would aid the patient, and the chest was closed. The postoperative course was not remarkable. The patient has since returned to school apparently unchanged from his preoperative state.

## SUMMARY

1. Several technics have been developed for performing closed or indirect intracardiac surgery with the surgeon being guided only by the feel of the instrument or the finger. Although effective for some cardiac defects, these procedures are not satisfactory for others.

2. The necessity for maintaining adequate metabolism during operative maneuvers inside the heart under direct vision with a bloodless field has resulted in investigations in three major fields concerning (a) the use of an extracorporeal heart or heart-lung mechanism, (b) combined mechanical pump-normal human-patient hook-up of Lillehei, and (c) general hypothermia.

3. The technical difficulties and risk associated with the use of the extracorporeal heart are great and have not yet been satisfactorily overcome.

4. Extensive experimental work on animals and humans during the last century and a half and recent clinical evidence indicate that general hypothermia permits direct surgery of the heart without excessive risk.

5. A satisfactory and proven technic for performing intracardiac surgery under direct vision in a bloodless field by the use of hypothermia is described.

6. The case reports of 4 patients with congenital heart disease operated upon during hypothermia are presented.

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## EDITORIAL

### EARLY REPAIR OF INGUINAL HERNIA

Inguinal hernia should be repaired earlier. There is no reason to observe an inguinal hernia in a normal baby until the baby grows up or until "it might go away".

Too many inguinal hernias become surgical emergencies in the first 6 months of life because of incarceration. It is not fair to create an anxious or neurotic mother with the admonition, "if the hernia gets caught, you must call me promptly." Babies are always crying and mothers are always looking and rightfully wondering if the hernia is "caught."

An elective herniorraphy in an infant is simple. The mortality of elective herniorraphy with good anesthesia is zero. A suggested time to consider operation would be when the birth weight has been regained. The mother of the baby is now happy and no longer worried.

John Bailey, M.D.

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